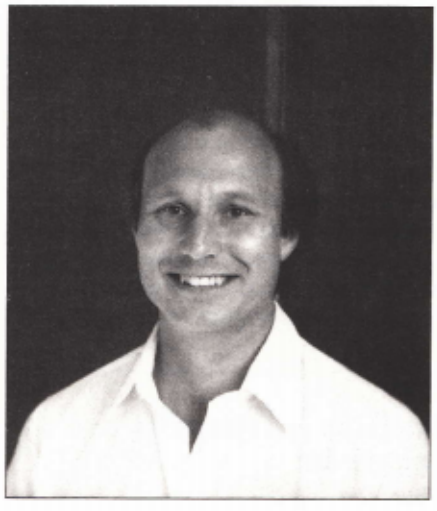
Headache: Towards an Integrated Understanding — RJ Henbest



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Curriculum vitae

Ronald J Henbest was born in Edmonton, Alberta (Canada) where he qualified in 1974 with a BSc in Maths and Psychology and in 1978 with an MD from the University of Alberta. He then completed two years postgraduate study (residency) in Family Medicine with the Department of Family Medicine at the University of Western Ontario (Canada) and obtained his CCFP from the College of Family Physicians of Canada. Ron joined the Department of Family Medicine at Medunsa in 1980. He has a particular interest in the doctor-patient interaction and its importance for healing. He returned to the University of Western Ontario in 1984 to take their Master of Clinical Science Degree in Family Medicine (MCISc), which emphasizes patient care, teaching and learning, and research. His thesis on Patient-Centred Care involved the development of a method for measuring patient-centredness and testing it against patient outcomes. In 1989, Ron returned to his home city, Edmonton, for a period of 21 months where he was engaged as an associated professor in the Department of Family Medicine at the University of Alberta. During this time, he also completed further training in systemic family therapy. In October 1990, Ron returned, with his wife Judy and four year old son Benji, this time as associate professor and deputy head of the Department of Family Medicine at Medunsa.

Summary

This paper describes a number of observations on the nature of headache, considers relevant data concerning the neuroanatomy and pathophysiology of headaches, theories of pain, and systems theory, and presents a classification of headache for use in family practice. There is now a great deal of interesting and useful information available, that if applied, can help make consultations with patients presenting with headaches, both more enjoyable and productive.

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The term "headache" is commonly employed to refer to almost any annoying problem - by doctors and patients alike. For the doctor who has little understanding of headache, the patient may *be*, not merely *have* a headache.

This article is the first in a series concerned with understanding headache and the people who present with them.

I shall begin with a number of observations on the nature of headache, discuss various data relevant to an understanding of headaches, including contributions from the fields of neuroanatomy and pathophysiology, pain theory and systems theory and then conclude by presenting a classification of headaches suitable for family practice.

Further articles will discuss the various categories and specific headache entities, describe an approach to the assessment of the patient based upon the classification, and present an integrated approach to management.

I would like to start with a statement that has stuck with me through the years, a statement made by Harold Wolff, an inspiring teacher and respected neurologist, neurosurgeon and scientist, in his classic work, called *Headache and Other Head Pain*. "Since the human animal prides himself on 'using his head', it is ironic and perhaps not without meaning that his head should be the source of so much discomfort."[1] Wolff went on to say that, "Though pain always means 'something wrong', with headache it most often means 'wrong direction' or 'wrong pace' - a biologic reprimand rather than a threat."[1]

Wolff's early statements have increasingly had their truth demonstrated in that the vast majority of headaches have been found to be due to readily reversible bodily changes and are related to the difficulties and frustrations of this life.

In contrast, the headaches due to brain tumor, brain abscesses, meningitis, subdural and subarachnoid haemorrhage, arteritis, and the pains of the major neuralgias make up only a small proportion of the total number of headaches.

A second observation made by Wolff was that headaches may be equally intense whether malignant or benign and he captured the dilemma of both doctor and patient in these words: "...there are few instances in human experience where so much pain may

mean so little in terms of tissue injury, failure to separate the ominous from the trivial may cost life or create paralyzing fears".[1]

Mankind's long experience with headache has provided much speculation about its nature and management. Headaches would seem to have begun with the beginning of man. Reference to headache is found in ancient mythology and as recently as the time of the Roman Empire, headaches were believed to be inflicted by the gods upon those who incurred their divine displeasure. A brief modern historical perspective is given by Friedman,[2] where he describes that in 1890, the common cause of headache was bad air; in 1910, it was focal infection; in 1920, it was refractive error; in 1930 it was sinusitis or allergy; in 1940, it was stretching the meninges or gallbladder disease; in 1950, the prime cause was tension headache; in the early 1970's, it was diet or depression and by the late 1970's it was thought to be an immunological disorder.

Headache often means wrong direction or wrong pace

I need hardly remind you that headache is a symptom, not a diagnosis. It is a symptom that is not specific of any disease and may be due to a broad range of problems ranging from structural to physiological, to psychological, or to spiritual or any combination of these. Thus we must deal with the patient, the person who presents with headaches, rather than merely with the symptom. I think that the aphorism that a correct assessment determines the management is especially true of the symptom of headache. Further I think that to effectively assess and manage a patient with headache, it is important not only to understand the basic neuroanatomy and pathophysiology of headache, but also to integrate the knowledge gained from general pain theory, and systems theory.

Neuroanatomy and Pathophysiology

Pain-Sensitive Structures

Even today, the basis of our knowledge of the pain-sensitive structures inside the skull comes from the original observations made by Ray and Wolff in 1940[3] from a series of 45 patients undergoing surgical procedures on the head. Their investigations showed that the following intracranial structures are pain sensitive: (1) the vessels (the great venous sinuses and their tributaries from the surface of the brain, the dural arteries, such as the anterior and middle meningeal arteries and the cerebral arteries at the base of the brain), (2) parts of the dura at the base of the brain, and (3) the nerves (the pain-sensitive fibres of the fifth, ninth and tenth cranial nerves and the upper cervical nerves). Of note, the cranium, the parenchyma of the brain, most of the dura, most of the pia-arachnoid, the ependymal lining of the ventricles, and the choroid plexuses are not sensitive to pain.

It was also noted that, except for those sensations that result from stimulation of the parenchyma and nerves, the only sensation that is experienced on stimulation of the intracranial structures is pain.

Remarkably, headache due to intracranial disease is referred pain.[3] Stimulation of structures on or above the superior surface of the tentorium cerebelli results in pain referred anteriorly to the intra-aural plane (ie the anterior two thirds of the head) by the fifth cranial nerve; whereas, stimulation of structures on or below the inferior surface of the tentorium cerebelli results in pain in various regions posterior to the intra-aural plane by the ninth and tenth cranial nerves and the upper three cervical nerves. Thus, in exception to the generalization that the site of headache closely overlies its intracranial origin, pain arising from the posterior half of the sagittal sinus or the upper surface of the transverse sinus lying partly under the occipital bone is referred forwards to the forehead of the same side.

Headache is a symptom, not a diagnosis

In contrast to the select intra-cranial structures that are pain-sensitive, virtually all of the extracranial structures, including the skin of the scalp, its blood supply and appendages, the blood vessels (especially the arteries) and the head and neck muscles, are pain sensitive.

Mechanisms of Head Pain

Our knowledge of the mechanisms for the production of intracranial head pain we also owe to Wolff and his colleagues.[3] The mechanisms are: (1) traction/displacement, (2) distension, (3) inflammation and (4) direct pressure. For example,

traction on the veins that pass to the sagittal and transverse sinuses from the cerebral cortex results in a dull, aching pain over a wide area on the front, top, and side of the head. Traction on the middle meningeal arteries causes headache as far forward as the eye and as far back as the ear, depending on whether the

Headache due to intracranial disease is referred pain

traction is primarily on the more anterior or the more posterior branches. Traction on the intracranial portion of the internal carotid arteries and the circle of Willis causes headache in the region of the eyes or in the front, top or sides of the head. Space occupying lesions such as tumors, brain abscesses, and haematomas may all cause pain through this mechanism.

Distention and dilatation of the intracranial arteries results in a throbbing or pounding headache. The arteries responsible for such pain are the pial arteries (chiefly at the base), and the dural arteries (especially the middle meningeal). Included in this category are headaches due to fever, sepsis, increased blood pressure, and many noxious substances.

Inflammation involving the pain sensitive structures at the base of the brain causes severe headache. In keeping with the referral patterns already described, the headache is chiefly over the occiput when the inflammation is limited to the posterior fossa; whereas, it is primarily frontal when the inflammation is in the supratentorial fossa. The headaches associated with meningitis, subarachnoid haemorrhage, or meningeal invasion by tumor are examples of headaches caused by inflammation.

Direct pressure by tumors on nerves possessing many pain-conducting fibres may cause pain in the distribution of the nerve involved. For example, compression of the intracranial portions of the ninth and tenth cranial nerves produces pain in and behind the corresponding ear, while compression of the upper cervical roots causes pain in the back of the head and neck.

Commonly, intracranial diseases cause headache through more than one of these mechanisms and by involvement of more than one pain-sensitive structure.

Having said the above, it is important to remember that the majority of headaches result from reversible tissue changes, that is, from: (1) sustained contraction of the musculoligamentous structures of the head and neck which when severe, may be enhanced by the presence of ischemia in the muscle due to vaso-constriction of the vessels to the contracted muscle.[1] (muscle contraction or tension headaches); (2) vascular dilatation of the intra- and extracranial blood vessels[3] associated with a sterile local inflammatory reaction[4] (headaches of the migraine type including classic and common migraine, hemiplegic and ophthalmoplegic migraine, and cluster headache) and (3) inflammation or distortion of extracranial structures (including those of the scalp, eyes, ears, nose, sinuses, teeth and neck.)

I think it is also important to keep in mind that head pain from any cause can arouse reflex contraction of the skeletal muscles of the head and neck which can greatly influence the patient's experience and description of the pain.

Theories of Pain

The traditional theory of pain is often referred to as "specificity theory"[5] and has been taken so much for granted in most medical schools and texts that it has, at least until recently, been considered fact rather than theory. Specificity theory states that a specific pain system transmits messages from pain receptors in the skin to a pain centre in the brain. Descartes is credited as having given the best classical description of the theory in 1664. He conceived of the pain system as a straight through channel from the skin to the brain analogous to the bell-ringing mechanism in a church: the rope is pulled at the bottom of the tower and the bell rings in the belfry. This

Most headaches result from reversable tissue changes

theory, with a number of additional developments persisted as a very powerful theory for some three hundred years.

Then, in 1965 Melzack and Wall[6] put forward a theory of pain that was to have a profound impact both on subsequent research and on the treatment of pain. The theory was called the gate-control theory and it proposed that a neural mechanism in the dorsal horns of the spinal cord

acts like a gate which can increase or decrease the flow of nerve impulses from peripheral fibres to the central nervous system. Thus, somatic signals can be modified and even blocked at the earliest stage of transmission in the nervous system. When the amount of stimulation passing through the gate exceeds a critical level, it activates the neural areas responsible for pain experience and response. By 1982 substantial advances in knowledge had led to modifications of the gate - control theory which was published in Melzack and Wall's fascinating book called, The Challenge of Pain.[5]

Melzack and Wall provide substantial evidence suggesting that the small cells of the substantia gelatinosa, (located in the dorsal horns of the spinal cord), act as the primary source of gate control. The substantia gelatinosa has been shown to contain both excitatory and inhibitory interneurons and a host of peptides including enkephalins and at least eight others.

In refuting the idea that pain is a simple sensation transmitted by a direct line to a pain centre, the major impact of the gate-control theory, initially, was to stimulate new thinking about pain. Later, it provided the conceptual framework for integrating the sensory, affective, and cognitive aspects of pain and in doing so, was in keeping with another important development that has gradually gained momentum during the latter half of this century.

Systems Theory

In medicine, the temptation has been great to look for the cause of a symptom as if there was only one, all inclusive, straight-forward cause for any given symptom. But, as has been increasingly recognized, symptoms are almost always the result of a number of factors, and the contextual and multifactorial view offered by systems theory has been very helpful in breaking the linear, specific cause and effect kind of thinking that has predominated for so long.

Table I. Classification of Headache

I. Common Functional Headaches

1. Tension/Muscle Contraction
2. Vascular Headaches of the Migraine Type
3. Psychogenic: No known mechanism
4. Combined

II. Rarer Organic headaches

1. Traction/Direct Pressure. Mass lesions: eg tumors, hematomas, abscesses
2. Dilatation of Cranial Arteries
   a) Toxic Vascular: eg febrile conditions, toxic substances, drug withdrawal, misc.
   b) Elevated Blood Pressure.
3. Inflammation eg meningitis, arteritis, subarachnoid haemorrhage
4. Conditions of the Cranial Nerves eg Neuritides and Neuralgias
5. Diseases of the Eyes, Ears, Nose, Mouth, Sinuses, Teeth or other Cranial or Neck Structures.

Crouch and Roberts have made a significant contribution to a broader understanding of symptoms in their challenging application of family systems theory to medical practice.[7] A systems understanding is especially important for symptoms such as headache, that may be indicative of a very broad range of problems. Crouch and Roberts provide numerous detailed examples of how a systems approach, leading to an understanding of the broader issues involved, results in better outcomes. Examples presented include the tension headaches in one member of a nice couple who have never had a cross word; the headaches of an 11-year old boy in a reconstituted family with over involved parents; and a 30 year old woman, with increasingly frequent migraine headaches, who feels isolated from her working husband and overwhelmed by the demands of caring for 3 small children.

Classification of Headache

Over the years, there have been a number of classifications of headache presented in the literature[4,8,13] but they can be considered variations on a theme, each with a different emphasis, rather than different entities. The basis for all of the classifications is that first proposed by Wolff and then published by the Ad Hoc Committee on Classification of Headache[8] (of which both Friedman and Wolff were prominent members). It is a comprehensive list based primarily on mechanisms and consists of 15 major categories. One variation of the theme is to emphasise primary versus secondary;[9] while another emphasises chronic recurring headaches versus acute headaches associated with pathology.[11]

... Headache

The classification presented in Table 1 is a modification that I have personally used for over a decade. There are just two main categories: I. Common Functional, and II. Rarer Organic. Each of these terms is significant. The common functional headaches are functional in the sense of "not being caused by structural changes" and are much commoner than those categorized as rarer organic headaches. In industrialized societies they account for some 80% of all headaches presented to family doctors.[14] Even in the busy general outpatient department at Ga-Rankuwa Hospital they accounted for 73% of the headaches presented by patients as their major complaint.[15]

The classification is based primarily on the known mechanisms of headache described earlier in this paper. As touched on briefly, the major mechanisms of pain are accompanied by fairly pattern-specific symptomatology (upon which the classic descriptions of the various headaches are based). These will be discussed in more detail in future papers. A few comments regarding the classification for purposes of clarification follow. The term psychogenic as used in this classification, does not refer to tension headaches, but rather to headaches of a bizarre description which cannot be readily explained in terms of known physiologic mechanisms (unlike tension and migraine headaches). Combined headaches refer to a combination of any of the other three common functional headaches.

The common functional headaches represent a continuum rather than distinct entities

The rarer organic group comprises a wide variety of disease entities that involve tissue pathology. Fortunately, the large majority of organic headaches are caused by relatively benign conditions. Of the five categories, by far the commonest are the vascular headaches sub-classified as the toxic vascular category, and diseases of the extracranial structures. The toxic vascular headaches are characterized by a generalized throbbing and include the headaches experienced as a result of (1) febrile conditions such as influenza and more strikingly, malaria and typhoid fever; (2) toxic substances (chemical headaches) including both pharmacologic agents such as the vasodilators, nitrates, indomethacin, estrogens, progesterone, reserpine and ergotamine tartrate, and nonpharmachologic substances such as carbon monoxide, lead, benzene, carbon tetrachloride, insecticides, smoking, and food additives; (3) withdrawal from drugs such as caffeine, ergot, any analgesic, phenothiazines, amphetamines, and alcohol and (4) miscellaneous conditions including hypoglycemia and hypoxia.

The other vascular headache category, that of increased blood pressure, is much less common and consists of 3 sub-categories that can be readily recognized by the context and description of the headache. These headaches are associated with: (1) a sudden major increase in the blood pressure such as may occur with violent exercise, anger, or sexual excitement, (2) severe essential hypertension (with a diastolic greater than 120 mmHg): and (3) renal failure, azotemia and increased intracranial pressure (hypertensive encephalopathy).

I shall conclude this section with three additional comments. The first is in response to the question, "Why bother with a classification?", followed by the statement, "After all, I know a few types of headache and can refer the rest." This classification is primarily a means of organizing information. What I have observed, is that patients are sometimes treated inappropriately if one has only a few headache entities in mind to treat. Perhaps the commonest examples I see are patients with recognisable, throbbing, vascular headaches being treated for migraine to little avail when their headaches are due to caffeine withdrawal. This classification helps prevent me from doing that. Vascular type headaches not typical for migraine do not need to be automatically labelled common migraine; one of the toxic vascular causes can be sought and managed appropriately.

The human animal prides himself on using his head - but this very head is the source of much discomfort

The second comment is a caution, that classification, by its very nature, is a generalising, and thus dehumanising process which ignores individual differences. It not only cannot replace, but may actually hinder understanding of the person and his or her unique illness.

... Headache

The final comment is in recognition of the growing understanding that the common functional headaches represent a continuum rather than distinct entities. A connection between muscle contraction and migraine headache that has long intrigued me has been the observation that both types of headache represent a response to a stressor (of one kind or another) and that the response is similar; that is, muscle contraction (skeletal muscle contraction in the case of muscle contraction headaches, vascular smooth muscle contraction as the initiating event for migraine headaches). Thus both may be seen as "tension" or "stress-induced" headaches in the broader sense of the terms. Although some would argue for a single category such as "recurrent non-specific headaches (RNSH), [14,16] I find it helpful to attempt to differentiate the headaches further if possible, especially in terms of identifying triggers or stressors that may have wonderful implications for preventive management (for example, there are certain triggers that I am more likely to look for if there is a significant vascular component to the headache). However, the recognition of this underlying continuum is most helpful in at least two ways. Firstly it removes some of the awful mystique of migraine for the patient, and secondly, it may help doctors understand the range of, and overlap of, symptoms encountered in assessing common functional headaches.

Conclusion

There is now a great deal of both interesting and useful information available that can help transform the consultation with a patient who presents with a headache into one of the more enjoyable and satisfying patient encounters that we, as doctors, may experience.

References

1. Wolff HG. Headache and Other Head Pain. 2nd ed. New York: Oxford University Press, 1963: Preface to the 1st ed (1948).
2. Friedman AP. Nature of Headache. Headache 1979; (April): 163-7.
3. Ray BS, Wolff HG. Experimental Studies on Headache: Pain Sensitive Structures of the Head and Their Significance in Headache. Arch Surg 1940; 41: 813-56.
4. Hansotia P. Evaluation and Treatment of Headache. Postgrad Med 1986; 79: 75-84.
5. Melzack R, Wall PD. The Challenge of Pain. New York: Penguin Books, 1982.
6. Melzack R, Wall PD. Pain Mechanisms: a New Theory. Science 1965; 150: 971-9.
7. Crouch MA, Roberts L. The Family in Medical Practice: A Family Systems Primer. London: Springer-Verlag, 1987.
8. Ad Hoc Committee on Classification of Headache. JAMA 1962; 179: 717-8.
9. Caviness VS, O'Brien P. Headache. N Engl J Med 1980; 302: 446-9.
10. Diamond S, Dalessio DJ. The Practising Physicians' Approach to Headache. 2nd ed. Baltmore: Williams and Wilkins, 1979.
11. Friedman AP. Current Concepts in the Diagnosis and Treatment of Chronic Recurring Headache. Med Clin North Am 1972; 56: 1257-71.
12. Kamin M. Headache. In Rakel RE, ed. Textbook of Family Practice. 4th ed. London: WB Saunders Co, 1990: 1427.
13. Headache Classification Committee of the International Society. Classification and Diagnostic Criteria for Headache Disorders. Cephalgia 1988; 8 Suppl. 7: 1-96.
14. McWhinney IR. A Textbook of Family Medicine. Oxford: Oxford University Press, 1989.
15. Roux JGH. A Descriptive Study of Headaches in the Outpatient Department, Ga-Rankuwa Hospital. S Afr Fam Pract 1984; 5: 283-9.
16. Headache Study Group of the University of Western Ontario. Predictors of Outcome in Headache Patients Presenting to Family Physicians - a One Year Prospective Study. Headache 1986; 26: 285-94.